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Case Report

Seizures and Unilateral Cystic Lesion of the Basal Ganglia: an Unusual Clinical and Radiological Manifestation of Chronic Non-fatal Carbon Monoxide (CO) Poisoning

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INTRODUCTION

Chronic non-fatal carbon monoxide (CO) poisoning is common and frequently unrecognized as the signs and symptoms are relatively non-specific [1]. We present a case of chronic non-fatal CO poisoning presenting with epilepsy in whom magnetic resonance imaging (MRI) of the brain showed a unilateral cystic lesion in the basal ganglia with bilateral dilatation of temporal horns suggestive of hippocampal atrophy. A history of CO inhalation and an awareness of possibility of varied and unusual clinical and imaging manifestations are important for recognition of non-fatal CO poisoning, especially when patients are in the chronic stage.

CASE REPORT

A 37-year-old woman presented with a history of seizures for 11 months. The seizures initially occurred once a month and later increased in frequency. During each episode she experienced feelings of de-personalization and de-realization such as daydreaming with unreal thoughts, which were unpleasant. There were no associated olfactory or gustatory hallucinations, epigastric aura, incontinence or tongue biting. These episodes sometimes proceeded to complete loss of consciousness followed by feeling unwell on regaining consciousness. The patient also had symptoms suggestive of orthostatic hypotension but the "tilt test" was negative. The patient gave a history of chronic CO exposure extending over a 7 year period starting from April 1989 during which she had persistent tiredness and headache associated with cognitive symptoms, personality changes and depression. Her house was found to be heavily contaminated with CO due to a faulty heating appliance.

On examination there were no abnormal neurological signs. Electroencephalogram (EEG) was unremarkable. MRI of the brain 5 years after the end of chronic non-fatal CO exposure showed a well-defined lesion of the globus pallidus on the left, which was pear-shaped on axial images (Fig. 1*b*, *c*), ellipsoid on sagittal image figure (Fig. 1*a*) and the signal intensity was that of cerebrospinal fluid (CSF) on T1-weighted, proton density and T2-weighted images (Fig. 1*a*, *b*, *c*). Appearances suggest a cystic lesion in left globus pallidus. There was also presence of bilateral mild dilatation of temporal horns (Fig. 2*a*, *b*) suggestive of hippocampal atrophy. There was no evidence of white matter lesions or hypointense lesions in the corpus striatum.

DISCUSSION

CO poisoning is the commonest cause of accidental poisoning in Europe and USA [2]. Clinical presentation is non-specific and ranges from nausea and headache to profound central nervous system (CNS) dysfunction [3]. The long-term sequel of chronic non-fatal poisoning has received scant attention [4,5]. In most patients the symptoms disappear after several months. However, Tvedt and Kjuus reported two cases of permanent symptoms after CO poisoning [5]. Seizures were generally regarded as a manifestation of extreme, generally near fatal CO poisoning [6]. Our patient's presentation of seizures due to chronic non-fatal CO poisoning is therefore unusual but not

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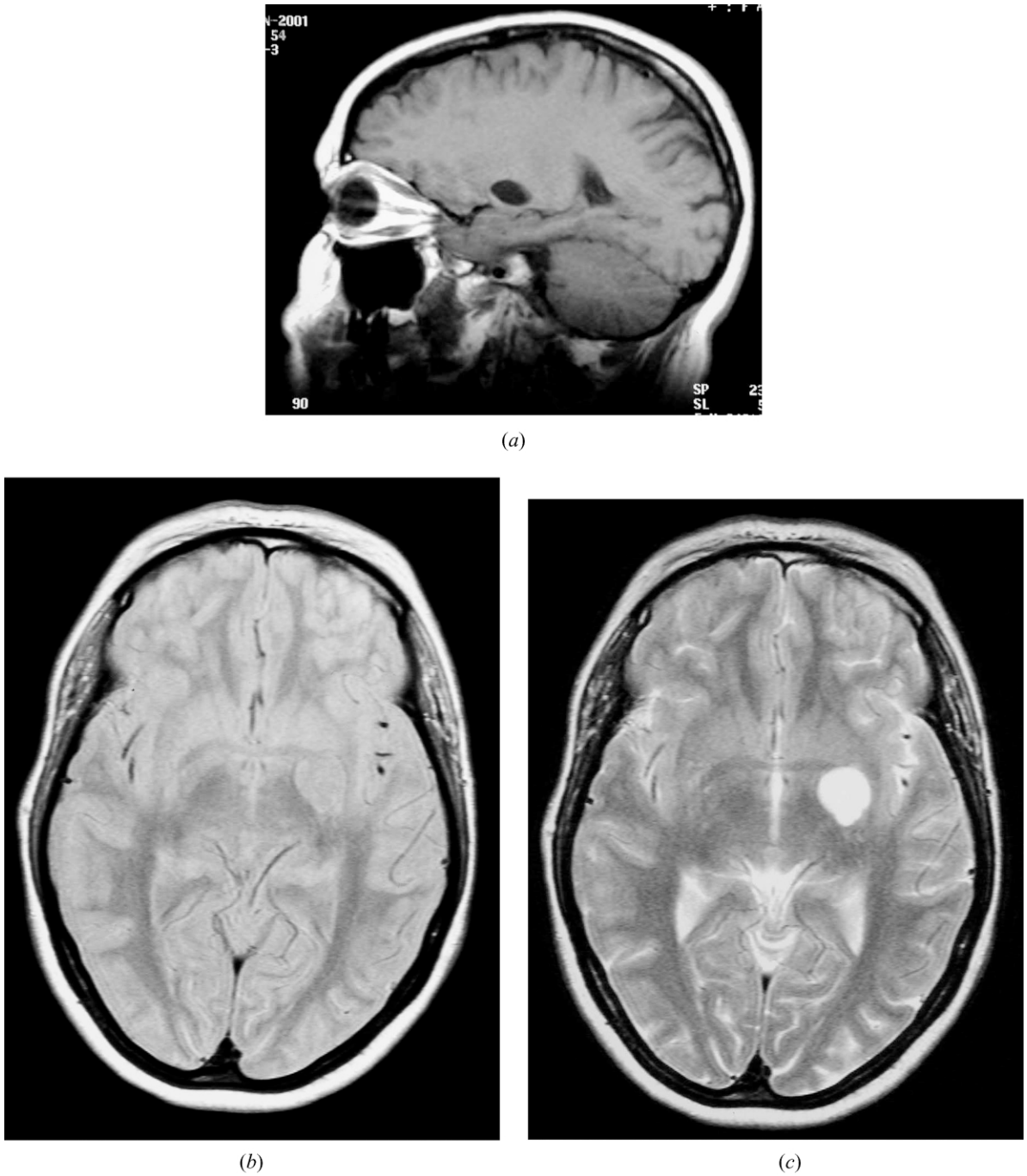


(*a*)

(*b*)

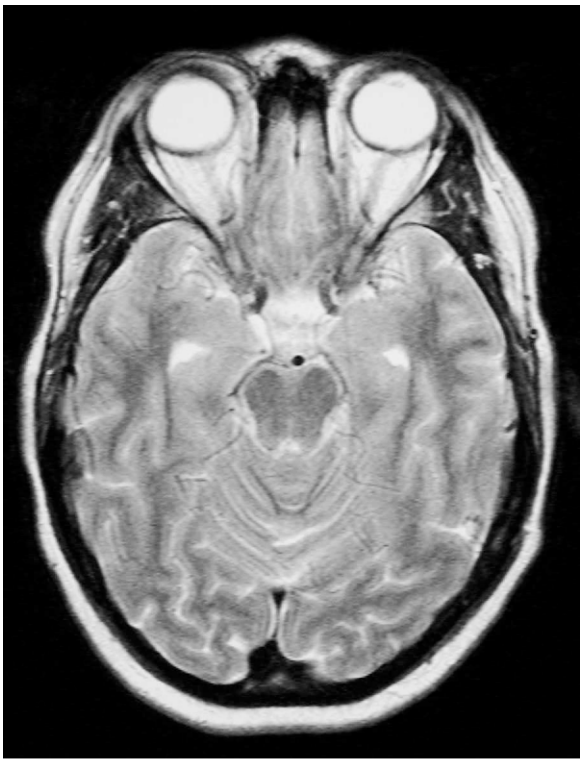
(*c*)

Fig. 1 – Sagittal T1-weighted image (*a*), axial proton density (*b*) and axial T2-weighted image (*c*) demonstrating a cystic lesion in the left basal ganglia region.

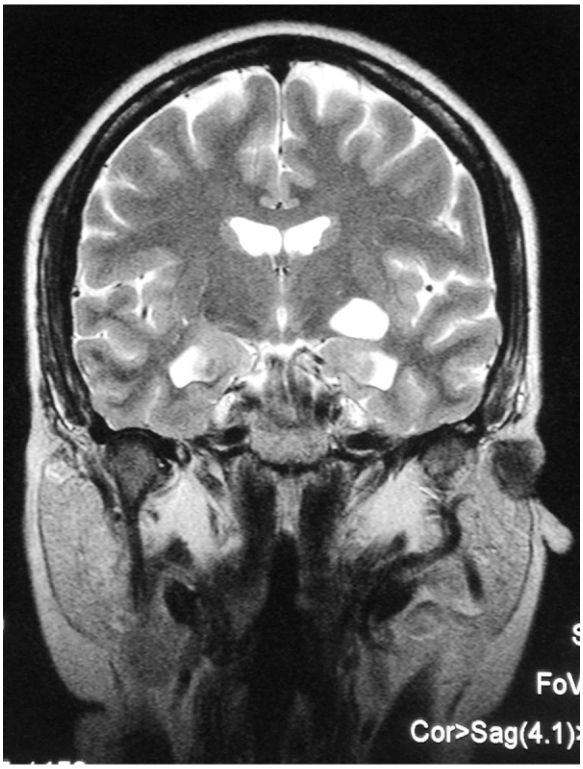
implausible, as no particular degree of carboxyhaemoglobin has been correlated with the presence of seizures [6].

The cerebral abnormalities in CO toxicity, as studied in post-mortem studies, were distributed bilaterally in basal ganglia, deep white matter, cerebral cortex and hippocampus [7]. Selective necrosis and degeneration of the globus pallidus are characteristic autopsy findings in patients with severe CO poisoning [8] although not specific [9]. Unilateral involvement of globus pallidus has also been reported in head trauma [10], post-encephalitis and post-meningitis lesions [11], basal ganglia tumours, stroke and Leigh syndrome [9]. The mechanisms for the predilection of globus pallidus is unclear but is suggested to be due to the hypotensive effects of CO poisoning in the watershed territory of arterial supply [12]. Symptoms relating to pallidal lesions are commonly observed after a time interval of days or months, and are those of cognitive impairment and personality changes [13].

MRI is very sensitive in detecting lesions of white matter and basal ganglia in delayed encephalopathies associated with CO poisoning [14,15]. Reports of MRI in CO poisoning are



(*a*)



(*b*)

Fig. 2 – (*a*) Axial T2-weighted image and (*b*) coronal T2-weighted image, demonstrating mild atrophy of both hippocampus.

sparse [16] and the majority of cases previously reported were in the acute and sub-acute stage. The characteristic findings on T2-weighted images are high-intensity lesions in the bilateral periventricular white matter and pallidal regions in the chronic stage [16–18]. High-resolution MRI findings consist of hippocampus atrophy with or without diffuse cortical atrophy [19]. Globus pallidus lesions in the chronic stage become more apparent [20] and in many cases do not correlate directly to clinical status and outcome, whereas the presence of diffuse white matter lesions which tend to diminish in the chronic stages [20] is a more reliable index of both [21]. Asymmetric and unilateral lesions were previously reported in the chronic stage [16,22] and could reflect a portion of the brain being potentially at high risk for hypoxia presumably due to patterns of arterial supply and higher metabolic rates [16].

At presentation the clinical diagnosis was complex partial seizure and an MRI was performed to detect temporal lobe lesions. On MRI an initial diagnosis of incidental arachnoid cyst of left globus pallidus was made. Review of MRI with clinical history of chronic non-fatal CO poisoning led to the diagnosis of cystic lesion of the left globus pallidus, which has not been reported in the literature previously. One criticism of our presentation is that we do not have pathological proof for the histology of the cystic lesion. Biopsy of incidentally discovered lesions is not automatically indicated, especially if the most likely cause is benign. The differential diagnoses of cystic lesion in the basal ganglia are giant Virchow Robin space, giant epithelial cyst, cystic lacunar infarct, and cystic forms of malacia [23]. Additionally mild dilatation of bilateral temporal horns suggestive of bilateral atrophy of hippocampus was also seen. It should be noted although the MRI study was obtained 5 years after the termination of chronic non-fatal CO poisoning, it is believed that the imaging findings represent lesions attributable to CO toxicity because the lesions are known to manifest as necrosis, one would expect them to be demonstrable for long periods [24]. Hence awareness of the potential for unilateral cystic pallidal lesion and unusual and varied clinical presentations would lead to more accurate MRI interpretation and hence have significant medicolegal implications in chronic non-fatal CO poisoning.

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